L Number	Hits	Search Text	DB	Time stamp
1	78	cln3	USPAT; US-PGPUB; DERWENT	2003/08/11 11:51
2	16	cln3 same (cancer$6 or prolifer$6 or overexpress$6 or upregulat$6)	USPAT; US-PGPUB; DERWENT	2003/08/11 11:53
3	5679	boustany-$.in. or guo-$.in. or amalfitano-$.in.	USPAT; US-PGPUB; DERWENT	2003/08/11 11:54
4	364	(boustany-$.in. or guo-$.in. or amalfitano-$.in.) and cancer	USPAT; US-PGPUB; DERWENT	2003/08/11 11:54
5	3	((boustany-$.in. or guo-$.in. or amalfitano-$.in.) and cancer) and cln3	USPAT; US-PGPUB; DERWENT	2003/08/11 11:54

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      (FILE 'HOME' ENTERED AT 12:35:55 ON 11 AUG 2003)

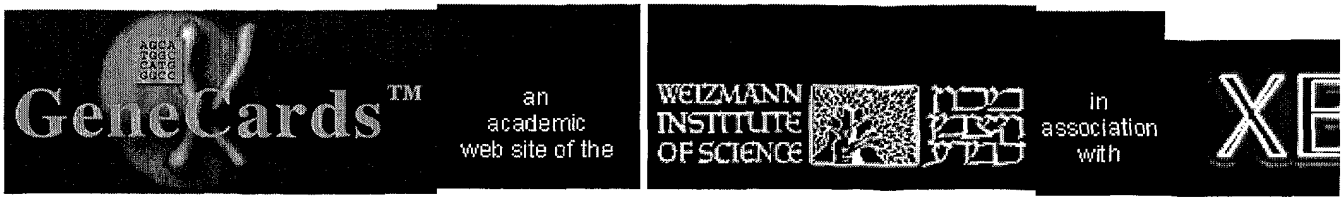
      FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 12:36:16 ON 11 AUG 2003
L1          1192 S CLN3
L2           118 S L1 AND OVEREXPRESS?
L3            17 S L2 AND (CANCER? OR PROLIFERATI?)
L4             7 DUP REM L3 (10 DUPLICATES REMOVED)
L5           205 S BOUSTANY R?/AU
                 E BOUSTANY R?/AU
L6            65 S E5-E6
L7            24 S L6 AND CLN3
L8            12 DUP REM L7 (12 DUPLICATES REMOVED)
                 E AMALIFTANO A?/AU

=>
```

L4 ANSWER 3 OF 7 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2001:109700 BIOSIS
DOCUMENT NUMBER: PREV200100109700
TITLE: Blocking of **CLN3** expression by antisense **CLN3** adenovirus suppresses **cancer** growth by modulating ceramide levels and delaying recovery from a nocodazole induced G2M block.
AUTHOR(S): Rylova, S. (1); Jansen, P.; Amalfitano, A.; Pane, M.; Boustany, R. M.
CORPORATE SOURCE: (1) Duke University Medical Center, Durham, NC USA
SOURCE: Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-766.2. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000 Society for Neuroscience
. ISSN: 0190-5295.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Juvenile Batten disease is a neurodegenerative disease. Accelerated apoptotic death of photoreceptors and neurons occurs due to defects in the **CLN3** gene. **CLN3** has antiapoptotic activity when **overexpressed** in NT2 neuronal precursor cells. We have shown **overexpression** of **CLN3** in a variety of **cancer** cell lines and solid colon **cancer** tissue: **CLN3** is **overexpressed** in glioblastoma (U-373G, T98g), neuroblastoma (IMR-32, SK-N-MC), prostate (Du145, PC-3, LNCap), breast (BT-20, BT-549, BT-474), colon (SW1116, SW480, HCT 116) and leukemia (HL-60) cell lines, but not in malignant melanoma or pancreatic **cancer** lines. An adenovirus bearing antisense **CLN3** (Ad-AsCLN3) was used to transduce BT-20, SW1116, T98g **cancer** cell lines and resulted in blocking of **CLN3** expression as seen by Western blot. Also suppression of **cancer** cell growth was seen by 3H-Thymidine incorporation and cell counting. Ceramide levels were increased 52% after transduction of DU145 prostate **cancer** cells with 40 MOI of Ad-AsCLN3 virus. Neuronal precursor NT2 stable cell lines both over and underexpressing **CLN3** were synchronized by blocking them at the G2/M phase of the cell cycle using Nocodazole. The cells **overexpressing** **CLN3** rapidly exited G2/M and proceeded through the cell cycle after removal of Nocodazole in comparison to NT2 cells underexpressing **CLN3** as seen by flow cytometry. Blocking of **CLN3** expression using Ad-AsCLN3 suppresses growth of **cancer** cells. This could be mediated by excess ceramide known to inhibit cell growth and result in cell cycle arrest.

L4 ANSWER 1 OF 7 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2002099667 MEDLINE
DOCUMENT NUMBER: 21818590 PubMed ID: 11830536
TITLE: The **CLN3** gene is a novel molecular target for **cancer** drug discovery.
AUTHOR: Rylova Svetlana N; Amalfitano Andrea; Persaud-Sawin Dixie-Ann; Guo Wei-Xing; Chang Jerry; Jansen Paul J; Proia Alan D; Boustany Rose-Mary
CORPORATE SOURCE: Department of Pediatrics, Duke University Medical Center, Durham, North Carolina 27710, USA.
CONTRACT NUMBER: R01 DK 52925 (NIDDK)
R02 NS 30170 (NINDS)
SOURCE: CANCER RESEARCH, (2002 Feb 1) 62 (3) 801-8.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200203
ENTRY DATE: Entered STN: 20020207
Last Updated on STN: 20020307
Entered Medline: 20020305

AB Juvenile Batten disease is a neurodegenerative disease caused by accelerated apoptotic death of photoreceptors and neurons attributable to defects in the **CLN3** gene. **CLN3** is antiapoptotic when **overexpressed** in NT2 neuronal precursor cells. **CLN3** negatively modulates endogenous ceramide levels in NT2 cells and acts upstream of ceramide generation. Because defects in regulation of apoptosis are involved in the development of **cancer**, we evaluated the expression of **CLN3** on both mRNA and protein levels in a variety of **cancer** cell lines and solid colon **cancer** tissue. We also observed the effect of the blocking of **CLN3** protein expression on **cancer** cell growth, survival, ceramide production, and apoptosis by using an adenovirus-bearing antisense **CLN3** construct. We show that **CLN3** mRNA and protein are **overexpressed** in glioblastoma (U-373G and T98g), neuroblastoma (IMR-32 and SK-N-MC), prostate (Du145, PC-3, and LNCaP), ovarian (SK-OV-3, SW626, and PA-1), breast (BT-20, BT-549, and BT-474), and colon (SW1116, SW480, and HCT 116) **cancer** cell lines but not in pancreatic (CAPAN and As-PC-1) or lung (A-549 and NCI-H520) **cancer** cell lines. **CLN3** is also up-regulated in mouse melanoma and breast carcinoma **cancer** cell lines. We found **CLN3** expression is 22-330% higher than in corresponding normal colon control tissue in 8 of 10 solid colon tumors. An adenovirus-expressing antisense **CLN3** (Ad-AS-**CLN3**) blocks **CLN3** protein expression in DU-145, BT-20, SW1116, and T98g **cancer** cell lines as seen by Western blot. Blocking of **CLN3** expression using Ad-AS-**CLN3** inhibits growth and viability of **cancer** cells. It also causes elevation in endogenous ceramide production through de novo ceramide synthesis and results in increased apoptosis as shown by propidium iodide and JC-1 staining. This suggests that Ad-AS-**CLN3** may be an option for therapy in some **cancers**. More importantly these results suggest that **CLN3** is a novel molecular target for **cancer** drug discovery.



SEARCH GeneCards
cln3
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RESULT:

1 GeneCard matches **your *precise* query** for **"cln3";**
It is represented by a minicard.
Click "Display" on the left to get the full GeneCard.

Display the complete GeneCard for this gene (**CLN3**)

More like this

Gene: CLN3 = ceroid-lipofuscinosis, neuronal 3, juvenile (Batten, Spielmeyer-Vogt disease) [Locus: 1

The following lines in the GeneCard text contribute to matching your query:

- GENE: **CLN3** (ceroid-lipofuscinosis, neuronal 3, juvenile (Batten, Spielmeyer-Vogt disease))
- OMIM: **CLN3** gene; **CLN3**, BTS | 607042 | Ceroid-lipofuscinosis, neuronal-3, juvenile
- MOUSE HOMOLOG: **Cln3** (on chromosome 7, 60.40 | Apr 08 2003 | gbaccs: AK078976 U47106 U6
NM_009907 cM)
- SWISSPROT: **CLN3** protein (Battenin) (Batten disease protein).
- SWISSPROT: GENE: **CLN3** OR BTS
- SWISSPROT: DISEASE: DEFECTS IN **CLN3** ARE A CAUSE OF BATTEN DISEASE (BD) (ALSO K
ONSET NEURONAL CEROID LIPOFUSCINOSIS TYPE 3; JNCL), A RECESSIVELY INHERITED NE
DISORDER OF CHILDHOOD CHARACTERIZED BY PROGRESSIVE LOSS OF VISION, SEIZURES
PSYCHOMOTOR DISTURBANCES. BIOCHEMICALLY, THE DISEASE IS CHARACTERIZED BY LY
ACCUMULATION OF HYDROPHOBIC MATERIAL, MAINLY ATP SYNTHASE SUBUNIT C. CLINICA
USUALLY FROM 5 TO 10 YEARS OF AGE. NO TREATMENT IS AVAILABLE AND BD IS USUALLY
DECADE. THE INCIDENCE IS ESTIMATED AT 1/20000 TO 1/100000 LIVE BIRTH, MAKING IT ONE
COMMON NEURODEGENERATIVE DISEASES OF CHILDHOOD.
- SWISSPROT: DATABASE: NCL **CLN3** -Neural Ceroid Lipofuscinoses mutation db.
- SWISSPROT: DATABASE: Mutations of the **CLN3** gene -Retina International's Scientific Newsletter
- HGMD: 120593 | HGMD entry for **CLN3** | mutations
- UNIGENE: Hs.194660 | ceroid-lipofuscinosis, neuronal 3, juvenile (Batten, Spielmeyer-Vogt disease
sapiens;Mar 24 2003 | **CLN3** | NM_000086 . . .

- LITERATURE: 7553855 | Isolation of a novel gene underlying Batten disease, **CLN3**. The Internatio
Consortium.|SP
- BLOCKS: PR01315, **CLN3** Batten's disease protein (battenin) signature;
- RZPD: id-**CLN3**
- euGenes: Organism=worm|WormSymbol=cln-3.1|WormLocation=V 11266193..11268210|WormDes
protein like|WormSimilarity=40%|WormLink=cln-3.1||Organism=worm|WormSymbol=cln-3.2|WormLo
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- HomoloGene: Organism=Rn|Symbol=|Location=
|Description=|Similarity=98.39|LocusLink=|Unigene=33154|GenBank=BE119371.1||Organism=Dr|Syr
|Description=|Similarity=71.77|LocusLink=|Unigene=104246|GenBank=BG303854.1||Organism=Mm|S

|Description=ceroid lipofuscinosis, neuronal 3, juvenile (Batten, Spielmeyer-Vogt disease)
|Similarity=80.54|LocusLink=12752|Unigene=232156|GenBank=NM_009907.1||Organism=Xl|Symbol
|Description=ESTs, Weakly similar to A57219 Batten disease-related protein **CLN3** - human [H.sapier
|Similarity=75.65|LocusLink=|Unigene=84969|GenBank=BJ043920.1||

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Developed at the Crown Human Genome Center & Bioinformatics Unit, at the Weizmann Institute of Science

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National Library of Medicine - Medical Subject Headings

2003 MeSH

MeSH Supplementary Concept Data

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Name of Substance	Batten disease protein CLN3
Record Type	C
Registry Number	0
Entry Term	CLN3 gene product, human
Entry Term	CLN3 protein, human
Entry Term	ceroid lipofuscinosis, neuronal 3 protein
Entry Term	CLN3 protein, Batten disease
Entry Term	battenin
Heading Mapped to	*Proteins
Indexing Information	Neuronal Ceroid-Lipofuscinosis
Source	Genomics 1997 Mar 1;40(2):346-50
Frequency	51
Note	base sequence in first source; GenBank U32680; mouse homolog = CLN3 PROTEIN, MOUSE; don't confuse with CLN3 PROTEIN, YEAST
Date of Entry	19970421
Revision Date	20030228
Unique ID	C105199

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